=> S CHROMOSOME (2A) (18) AND (BIPOLAR OR MANIC)

6999 CHROMOSOME

1649693 18

126 CHROMOSOME (2A) (18)

37700 BIPOLAR

414 MANIC

2 CHROMOSOME (2A) (18) AND (BIPOLAR OR MANIC)

=> D L1 1-2 CIT, AB

1. 5,914,394 Jun. 22, 1999 Methods and compositions for the diagnosis and treatment of neuropsychiatric disorders; Hong Chen, et al., 536/23.5; 435/69.1, 70.2, 91.1, 252.3, 320.1, 325, 333; 536/23.1, 24.1 [IMAGE AVAILABLE]

US PAT NO:

1.1

5,914,394 [IMAGE AVAILABLE]

L1: 1 of 2

ABSTRACT:

The present invention relates to the mammalian fsh16 gene, a novel gene associated with bipolar affective disorder (BAD) in humans. The invention encompasses fsh16 nucleic acids, recombinant DNA molecules, cloned genes or degenerate variants thereof, fsh16 gene products and antibodies directed against such gene products, cloning vectors containing mammalian fsh16 gene molecules, and hosts that have been genetically engineered to express such molecules. The invention further relates to methods for the identification of compounds that modulate the expression of fsh16 and to using such compounds as therapeutic agents in the treatment of fsh16 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh16 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compositions for the treatment these disorders.

2. 5,866,412, Feb. 2, 1999, **Chromosome 18** marker; Hong Chen, et al., 435/320.1, 243, 325; 536/23.1, 23.5 [IMAGE AVAILABLE]

US PAT NO:

5,866,412 [IMAGE AVAILABLE]

L1: 2 of 2

ABSTRACT:

The present invention relates to the mammalian fsh15w6 gene, a novel gene associated with bipolar affective disorder (BAD) in humans. The invention encompasses fsh15w6 nucleic acids, recombinant DNA molecules, cloned genes or degenerate variants thereof, fsh15w6 gene products and antibodies directed against such gene products, cloning vectors containing mammalian fsh15w6 gene molecules, and hosts that have been genetically engineered to express such molecules. The invention further relates to methods for the identification of compounds that modulate the expression of fsh15w6 and to using such compounds as therapeutic agents in the treatment of fsh15w6 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh15w6 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compositions for the treatment these disorders.

ANSWER 1 OF 6 MEDLINE L2 MEDLINE 1999264248 AN Assessing the feasibility of linkage disequilibrium methods for mapping DN complex traits: an initial screen for bipolar disorder TΙ Escamilla M A; McInnes L A; Spesny M; Reus V I; Service S K; Shimayoshi loci on chromosome 18. ΑU Tyler D J; Silva S; Molina J; Gallegos A; Meza L; Cruz M L; Batki S; N: Vinogradov S; Neylan T; Nguyen J B; Fournier E; Araya C; Barondes S H; Leon P; Sandkuijl L A; Freimer N B Neurogenetics Laboratory, University of California San Francisco, San CS Francisco, USA. MH00916 (NIMH) NC MH49499 (NIMH) MH48695 (NIMH) AMERICAN JOURNAL OF HUMAN GENETICS, (1999 Jun) 64 (6) 1670-8. SO Journal code: 3IM. ISSN: 0002-9297. United States CY Journal; Article; (JOURNAL ARTICLE) ידים English LA Priority Journals 199908 EMLinkage disequilibrium (LD) analysis has been promoted as a method of EW mapping disease genes, particularly in isolated populations, but has not ΑB yet been used for genome-screening studies of complex disorders. We present results of a study to investigate the feasibility of LD methods for genome screening using a sample of individuals affected with severe bipolar mood disorder (BP-I), from an isolated population of the Costa Rican central valley. Forty-eight patients with BP-I were genotyped for markers spaced at approximately 6-cM intervals across chromosome 18. Chromosome 18 was chosen because a previous genome-screening linkage study of two Costa Rican families had suggested a BP-I locus on this chromosome. Results of the current study suggest that LD methods will be useful for mapping BP-I in a larger sample. The results also support previously reported possible localizations (obtained from a separate collection of patients) of BP-I-susceptibility genes at two distinct sites on this chromosome. Current limitations of LD screening for identifying loci for complex traits are discussed, and recommendations are made for future research with these methods. ANSWER 2 OF 6 MEDLINE T₁2 1998351040 MEDLINE AΝ 98351040 Affective disorder associated with a balanced translocation DMTIinvolving chromosome 14 and 18. Overhauser J; Berrettini W H; Rojas K Department of Biochemistry and Molecular Pharmacology, Thomas Jefferson ΔIJ University, Philadelphia, PA 19107, USA. J_Overhauser@lac.jci.tju.edu PSYCHIATRIC GENETICS, (1998 Summer) 8 (2) 53-6. CS SO Journal code: B3X. ISSN: 0955-8829. ENGLAND: United Kingdom CYJournal; Article; (JOURNAL ARTICLE) DT

English

199901

Priority Journals

T.A

FS

EM

EW

We report a case of a women with psychiatric illness that includes bipolar disorder who has a karyotype of 46, XX, t(14;18) (q11.2;q22.1). The region on chromosome 18 that is involved in the translocation has been implicated in other families through linkage and association studies as possibly containing a gene for bipolar illness.

ANSWER 3 OF 6 MEDLINE L2

MEDLINE 97480722 AΝ

DN

Genomic structure and chromosomal localization of a human myo-inositol TImonophosphatase gene (IMPA).

Sjoholt G; Molven A; Lovlie R; Wilcox A; Sikela J M; Steen V M ΑU

Dr. Einar Martens' Research Group for Biological Psychiatry, Center for Molecular Medicine, Haukeland University Hospital, Bergen, Norway. CS

GENOMICS, (1997 oct 1) 45 (1) 113-22. Journal code: GEN. ISSN: 0888-7543.

United States CY

Journal; Article; (JOURNAL ARTICLE) DT

English LΑ

Priority Journals FS

GENBANK-Y11360; GENBANK-Y11361; GENBANK-Y11362; GENBANK-Y11363; OS GENBANK-Y11364; GENBANK-Y11365; GENBANK-Y11366; GENBANK-Y11367

199801 EM

19980104 EW

Manic-depressive illness is a serious psychiatric disorder that in many, but far from all, patients can be treated with lithium. The main AB causes for discontinuation of lithium therapy are unpleasant or serious side effects and lack of response. The reason for the striking variation in clinical efficacy of lithium treatment among bipolar patients is not known. The enzyme myo-inositol monophosphatase (IMPase) has been postulated as a target for the mood-stabilizing effects of lithium, but variation in the coding region of the human IMPA gene encoding IMPase activity has not been observed in manic-depressive patients (Steen et al., Pharmacogenetics, 1996, 6, 113-116). It is nevertheless conceivable that polymorphisms or mutations in the noncoding regions of this gene could influence the lithium response in psychiatric patients. As a first step in investigating this possibility, we here report the genomic structure of the human IMPA gene. The gene is composed of at least nine exons and covers more than 20 kb of sequence on chromosome 8q21.13-q21.3. In the 3'-untranslated part of the gene, we observed a polymorphism (a G to A transition) and also two short

similar to the inositol/cholin-responsive element consensus. Finally, we postulate that two additional IMPA-like transcripts originate from the human genome, one from a position close to IMPA itself on chromosome 8

and

the other from chromosome 18p. Our data may contribute to the identification of genetic factors involved in the pathogenesis and determination of treatment response in manic-depressive illness.

ANSWER 4 OF 6 MEDLINE T.2

MEDLINE 97430985 ΑN

97430985 DN

Lack of evidence for a major locus for bipolar disorder TΤ in the pericentromeric region of chromosome 18 in Irish pedigrees.

Mynett-Johnson L A; Murphy V E; Manley P; Shields D C; McKeon P ΑIJ

Department of Genetics, Trinity College Dublin, Ireland. CS

BIOLOGICAL PSYCHIATRY, (1997 Sep 15) 42 (6) 486-94. Journal code: A3S. ISSN: 0006-3223.

United States CY

(CLINICAL TRIAL) DT Journal; Article; (JOURNAL ARTICLE)

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English
LΑ
     Priority Journals
FS
     199712
EΜ
    Seven families, multiply affected by bipolar mood
     19971204
EW
     disorder, have been collected from the Irish population and have
AB
     been genotyped with microsatellite markers from the pericentromeric
     of chromosome 18, a region that has been implicated as
     a site for a susceptibility gene for this relative common psychiatric
     disorder. The families significantly excluded linkage of
     bipolar disorder to this region under various models.
     Although the data provided no evidence of linkage heterogeneity among
     families, the number of families investigated may be too small to exclude
     completely the possibility of linkage in a small number of families.
     ANSWER 5 OF 6 MEDLINE
T<sub>1</sub>2
                  MEDLINE
     97372982
ΑN
      97372982
     Cytogenetic abnormalities on chromosome 18 associated
DN
     with bipolar affective disorder or schizophrenia.
TΙ
     Mors O; Ewald H; Blackwood D; Muir W
     Institute for Basic Psychiatric Research, Risskov, Denmark.
ΑU
     BRITISH JOURNAL OF PSYCHIATRY, (1997 Mar) 170 278-80.
 SO
      Journal code: B1K. ISSN: 0007-1250.
      ENGLAND: United Kingdom
 CY
      Journal; Article; (JOURNAL ARTICLE)
 DT
      English
 LΑ
      Priority Journals
 FS
      BACKGROUND: A few recent linkage studies have shown a possible locus for
 EΜ
 AB
      bipolar disorder on chromosome 18.
      Cytogenetic studies may assist in the further localisation of
      susceptibility loci on this chromosome. METHOD: A search was made for
      abnormalities of chromosome 18 in two separate large
      cytogenetic databases. In Denmark detection of mental illness in subjects
      with chromosome abnormalities was done by cross-linking the two separate
      register of psychiatric and chromosome disorders. In Scotland the
      Cytogenetic Registry of the MRC Human Genetics Unit undertakes long-term
      clinical follow-up of all cases with chromosome abnormalities. RESULTS:
      Cross-linking the two Danish register's revealed a family with the rare
       karyotype abnormality inv(18) (p11.3;q21.1) with one inversion carrier
       also suffered from bipolar disorder. In this family
  who
       there were two other cases of bipolar disorder, but
       the karyotype of these cases could not be established. One family in
       Scotland showed a case of schizophrenia in a carrier of inv(18) with the
       same breakpoints as the Danish family. CONCLUSIONS: We suggest further
       studies of the 18p11.3 and 18q21.1 regions in order to identify genes
       involved in bipolar affective disorder and
       schizophrenia.
       ANSWER 6 OF 6 MEDLINE
  L2
                    MEDLINE
       97209418
  AN
       97209418
       Genetics of manic depressive illness.
  TΤ
       MacKinnon D F; Jamison K R; DePaulo J R
       Department of Psychiatry, Johns Hopkins University School of Medicine,
  ΑU
  CS
       Baltimore, Maryland 21287, USA.
       ANNUAL REVIEW OF NEUROSCIENCE, (1997) 20 355-73. Ref: 65
       Journal code: 5Z5. ISSN: 0147-006X.
       United States
  CY
       Journal; Article; (JOURNAL ARTICLE)
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TTC

General Review; (REVIEW) (REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199708

AB Manic depressive illness is a common and frequently debilitating familial psychiatric disorder. Efforts to understand the mechanisms of inheritance have been hindered by the complexity of the phenotype, which may range from benign mood swings to chronic psychosis, and by apparently nonmendelian modes of transmission. Early reports of linkage

to

chromosomal loci have fallen into doubt; however they have helped encourage the development of more sophisticated methods for analyzing complex phenotypes. Using such methods, linkage of manic depressive illness to loci on chromosome 18 has been reported and apparently replicated, and work is proceeding to identify genes associated

with what is probably a genetically heterogeneous set of disorders. As molecular mechanisms of inheritance are elucidated, it will be important to consider the ethical implications of genetic testing in a clinically and genetically complex disorder such as manic depressive illness.

ANSWER 1 OF 4 MEDLINE **L**6 MEDLINE 1998019047 NALinkage analysis of manic depression (bipolar 98019047 DN affective disorder) in Icelandic and British kindreds using markers on TIt.he Kalsi G; Smyth C; Brynjolfsson J; Sherrington R S; O'Neill J; Curtis D; Rifkin L; Murphy P; Petursson H; Gurling H M ΆU Molecular Psychiatry Laboratory, University College London Medical CS School, HUMAN HEREDITY, (1997 Sep-Oct) 47 (5) 268-78. SO Journal code: GE9. ISSN: 0001-5652. Switzerland CY Journal; Article; (JOURNAL ARTICLE) DTEnglish LAPriority Journals FS 199802 EMAttempts were made to follow up results of a previous linkage study which EWsuggested that a locus-modifying susceptibility to bipolar and related AB unipolar affective disorder might be present in the pericentromeric of the short arm of chromosome 18. Twenty-three region multiply affected pedigrees collected from Iceland and the UK were genotyped using three highly polymorphic microsatellite markers at D18S40 and D18S44 which span the region implicated. Lod score analyses D18S37, under the assumption of heterogeneity and non-parametric linkage analyses were performed. The total lod scores obtained were strongly negative, and analysis allowing for heterogeneity did not suggest that any subgroup of the families was linked. Model-free linkage analysis using extended relative pair analysis and MFLINK also failed to detect any evidence for linkage. Our study provides no support for the presence of a locus-modifying genetic susceptibility to bipolar affective disorder in the pericentromeric region of chromosome 18q11. Further analyses in independent samples should help to reveal whether our negative results are due to locus heterogeneity or whether the original results were false-positive. ANSWER 2 OF 4 MEDLINE 1.6 MEDLINE 96304711 NAMaternal inheritance and chromosome 18 allele sharing 96304711 DNΤI in unilineal bipolar illness pedigrees. Gershon E S; Badner J A; Detera-Wadleigh S D; Ferraro T N; Berrettini W H National Institute of Mental Health, Bethesda, Maryland 20892-1274, USA. ΔIJ CS AMERICAN JOURNAL OF MEDICAL GENETICS, (1996 Apr 9) 67 (2) 202-7. NC SO Journal code: 3L4. ISSN: 0148-7299. United States CY Journal; Article; (JOURNAL ARTICLE) DTEnglish LA Priority Journals FS We have replicated the observation of McMahon et al. [1995] that there is EMexcess maternal transmission of illness in a series of previously described unilineal Bipolar manic-depressive illness

extended pedigrees [Berrettini et al., 1991]. ("Transmission" is defined for any ill person in a pedigree when father or mother has a personal or immediate family history of major affective disorder.) We divided our pedigrees into exclusively maternal transmission (Mat) and mixed maternal-paternal transmission (in different pedigree branches) (Pat). Using affected sib-pair-analysis, linkage to a series of markers on chromosome 18p-cen was observed in the Pat but not the Mat pedigrees, with significantly greater identity by descent (IBD) at these markers in the Pat pedigrees. As compared with the pedigree series as a whole, the proportion of alleles IBD in the linkage region is much increased in the Pat pedigrees. As shown by Kruglyak and Lander [1995],

as

the sharing proportion of alleles in affected relative pairs increases, the number of such pairs needed to resolve the linkage region to a 1 cM interval becomes smaller. Genetic subdivision of an illness by clinical

or

pedigree configuration criteria may thus play an important role in discovery of disease susceptibility mutations.

- L6 ANSWER 3 OF 4 MEDLINE
- AN 96301288 MEDLINE
- DN 96301288
- TI Analysis of chromosome 18 DNA markers in multiplex pedigrees with manic depression.
- AU Coon H; Hoff M; Holik J; Hadley D; Fang N; Reimherr F; Wender P; Byerley W
- CS Department of Psychiatry, University of Utah Medical School, Salt Lake City 84121, USA.
- NC MH-44212 (NIMH) MH10168-F32 (NIMH) MO1-RR00064 (NCRR)
- HOI-RROUGGY (NCRR)
 +
 SO BIOLOGICAL PSYCHIATRY, (1996 Apr 15) 39 (8) 689-96.
- Journal code: A3S. ISSN: 0006-3223.
- CY United States
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals
- EM 199612
- AB Six pedigrees segregating manic-depressive illness (MDI) were analyzed for linkage to 21 highly polymorphic microsatellite DNA markers on chromosome 18. These markers span almost the entire length of the chromosome, and gaps between markers are less than 20 cM. In particular, we analyzed several markers localizing to the pericentromeric region of chromosome 18 which generated lod scores suggestive of linkage in an independent study. Lod score analysis was performed and results were examined by family. One region produced positive lod scores, though at 18q23 and not in the pericentromeric region. We additionally used two nonparametric

methods

because the true mode of transmission of MDI is unknown; results were again somewhat suggestive for markers in the region of 18q23 but not in the pericentromeric region.

- L6 ANSWER 4 OF 4 MEDLINE
- AN 94286549 MEDLINE
- DN 94286549
- TI Chromosome 18 DNA markers and manicdepressive illness: evidence for a susceptibility gene.
- AU Berrettini W H; Ferraro T N; Goldin L R; Weeks D E; Detera-Wadleigh S; Nurnberger J I Jr; Gershon E S
- CS Department of Psychiatry and Human Behavior, Jefferson Medical College, Thomas Jefferson University, Philadelphia, PA 19107.
- NC 1 P41 RR03655 (NCRR)
- SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF

AMERICA, (1994 Jun 21) 91 (13) 5918-21. Journal code: PV3. ISSN: 0027-8424.

- CY United States
- DT Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals; Cancer Journals
- EM 199409
 - In the course of a systematic genomic survey, 22 manicdepressive (bipolar) families were examined for linkage to 11 chromosome 18 pericentromeric marker loci, under dominant and recessive models. Overall logarithm of odds score analysis for the pedigree series was not significant under either model, but several families yielded logarithm of odds scores consistent with linkage under dominant or recessive models. Affected sibling pair analysis of these data yielded evidence for linkage (P < 0.001) at D18S21. Affected pedigree member analysis also suggests linkage, with multilocus results for five loci giving P < 0.0001 and P = 0.0007 for weighting functions f(p) = 1 and 1/square root p, respectively, where p is the allele frequency. These results imply a susceptibility gene in the pericentromeric region of chromosome 18, with a complex mode of inheritance. Two plausible candidate genes, a corticotropin receptor and the alpha subunit of a GTP binding protein, have been localized to this region.

=> d his

L1

(FILE 'HOME' ENTERED AT 14:29:09 ON 13 JUL 1999)

FILE 'MEDLINE' ENTERED AT 14:29:25 ON 13 JUL 1999

- 1488 S BIPOLAR AND MOOD AND DISORDER
- L2 6 S L1 AND CHROMOSOME (2A) (18?)
- L3 153 S MANIC AND DEPRESS? AND (18?)
- L4 1655 S CHROMOSOME (4A)(18?)
- L5 6 S L4 AND L3
- L6 4 S L5 NOT L2

=> s 18 and (bipolar or manic)

18536 BIPOLAR 1039 MANIC

L9 21 L8 AND (BIPOLAR OR MANIC)

=> d 19 1-21 bib, ab

L9 ANSWER 1 OF 21 CA COPYRIGHT 1999 ACS

AN 131:1252 CA

TI CCG repeats in cDNAs from human brain

AU Kleiderlein, John J.; Nisson, Paul E.; Jessee, Joel; Li, W.-B.; Becker,

Κ.

G.; Derby, Michael L.; Ross, Christopher A.; Margolis, R. L.

CS Department of Psychiatry, Division of Neurobiology, The Laboratories of Genetic Neurobiology and Molecular Neurobiology, Johns Hopkins University School of Medicine, Baltimore, MD, 21287, USA

SO Hum. Genet. (1998), 103(6), 666-673 CODEN: HUGEDQ; ISSN: 0340-6717

PB Springer-Verlag

DT Journal

LA English

Expansion mutations of trinucleotide repeats and other units of unstable DNA have been proposed to account for at least some of the genetic susceptibility to a no. of neuropsychiatric disorders, including bipolar affective disorder, schizophrenia, autism, and panic disorder. To generate addnl. candidate genes for these and other disorders, cDNA libraries from human brain were probed at high stringency for clones contg. CCG, CGC, GCC, CGG, GCG, and GGC repeats (referred to collectively as CCG repeats). Some 18 cDNAs contg. previously

unpublished or uncharacterized repeats were characterized for chromosomal locus, repeat length polymorphism, and similarity to genes of known function. The cDNAs were also compared with the 37 human genes with eight or more consecutive CCG triplets in GenBank. The repeats were mapped to a no. of loci, including 1p34, 2p11.2, 2q30-32, 3p21, 3p22, 4q35, 6q22, 7qter, 13p13, 17q24, (8p11) 19p13.3, 20q12, 20q13.3, and 22q12. Length polymorphism was detected in 50% of the repeats. The newly cloned cDNAs

include a complete transcript of human neurexin-1B, a portion of BCNG-1

newly described brain-specific ion channel), a previously unreported polymorphic repeat located in the 5' UTR region of the guanine nucleotide-binding protein (G-protein) .beta.2 subunit, and a human version of the mouse proline-rich protein 7. This list of cDNAs should expedite the search for expansion mutations assocd. With diseases of the central nervous system.

L9 ANSWER 2 OF 21 CA COPYRIGHT 1999 ACS

AN 130:178369 CA

TI ZGGBP1 proteins related to bipolar affective disorder type 1

IN Flannery, Angela Veronica; Finnegan, Maria Christina Martina

PA Zeneca Limited, UK

SO PCT Int. Appl., 58 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO. KIND DATE

APPLICATION NO. DATE

WO 98-GB2259 19980728 19990211 WO 9906539 A1 РΤ W: JP, US RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE 19970801 PRAI GB 97-16162 A new human gene (ZGGBP1) is described which is assocd. with neurol. affective disorders such as bipolar affective disorder. A full-length cDNA encoding human ZGGBP1 and a partial cDNA encoding murine ZGGBP1 are disclosed. Polymorphic variants of the gene and functional domains encoded within the gene are also provided. The gene maps to chromosome 18q21 and shows appreciable sequence homol. to the ned-4 gene on chromosome 15. The invention further relates to methods for identifying compds. which modulate the activity of ZGGBP1 protein, and to diagnostic assays for the detection of ZGGBP1 in biol. samples. ANSWER 3 OF 21 CA COPYRIGHT 1999 ACS Ь9 130:172974 CA ΑN Use of fsh05 gene and protein for the diagnosis and treatment of TТ neuropsychiatric disorders Chen, Hong; Freimer, Nelson B. ΙN Millennium Pharmaceuticals, Inc., USA; The Regents of the University of PA California PCT Int. Appl., 117 pp. SO CODEN: PIXXD2 DTPatent English LA FAN.CNT 1 APPLICATION NO. DATE KIND DATE PATENT NO. ____ -----19980722 19990204 WO 98-US15183 A1WO 9904825 PΙ W: AU, CA, JP RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, 19990216 AU 98-85805 19980722 AU 9885805 A1PRAI US 97-898082 19970722 WO 98-US15183 19980722 The present invention relates to the mammalian fsh05 gene, a novel gene AB assocd. with bipolar affective disorder (BAD) in humans. The invention encompasses fsh05 nucleic acids, recombinant DNA mols., cloned genes or degenerate variants thereof, fsh05 gene products and antibodies directed against such gene product's, cloning vectors contg. mammalian fsh05 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh05 and to using such compds. as therapeutic agents in the treatment of fsh05 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh05 disorders and neuropsychiatric disorders including schizophrenia,

attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compns. for the treatment of these disorders.

ANSWER 4 OF 21 CA COPYRIGHT 1999 ACS L9

129:340353 CA ΑN

No evidence for significant linkage between bipolar affective TI disorder and chromosome 18 pericentromeric markers in a large series of multiplex extended pedigrees

Knowles, James A.; Rao, Peter A.; Cox-Matise, Tara; Loth, Jo Ellen; De ΑU Jesus, Gracielle M.; Levine, Laura; Das, Kamna; Penchaszadeh, Graciela

K.; Alexander, Joyce R.; Lerer, Bernard; Endicott, Jean; Ott, Jurg; Gilliam, T. Conrad; Baron, Miron

Columbia University College of Physicians and Surgeons and New York State CS

Psychiatric Institute, Rockefeller University, New York, NY, 10032, USA Am. J. Hum. Genet. (1998), 62(4), 916-924 SO CODEN: AJHGAG; ISSN: 0002-9297 PBUniversity of Chicago Press DT Journal LAEnglish Bipolar affective disorder (BP) is a major neuropsychiatric AΒ disorder with high heritability and complex inheritance. Previously reported linkage between BP and DNA markers in the pericentromeric region of chromosome 18, with a parent-of-origin effect (linkage was present in pedigrees with paternal transmission and absent in pedigrees with exclusive maternal inheritance), has been a focus of interest in human genetics. We reexamd. the evidence in one of the largest samples reported to date (1013 genotyped individuals in 53 unilineal multiplex pedigrees), using 10 highly polymorphic markers and a range of parametric and nonparametric analyses. There was no evidence for significant linkage between BP and chromosome 18 pericentromeric markers in the sample as a whole, nor was there evidence for significant parent-of-origin effect (pedigrees with paternal transmission were not differentially linked to the implicated chromosomal region). Two-point LOD scores and single-locus sib-pair results gave some support for suggestive linkage, but this was not substantiated by multilocus anal., and the results were further tempered by multiple test effects. We conclude that there is no compelling evidence for linkage between BP and chromosome 18 pericentromeric markers in this sample. ANSWER 5 OF 21 CA COPYRIGHT 1999 ACS T.9 129:287565 CA TT Methods and compositions for the diagnosis and treatment of neuropsychiatric disorders ΙN Chen, Hong; Freimer, Nelson B. Millenium Pharmaceuticals, Inc., USA; The Regents of the University of PACalifornia PCT Int. Appl., 94 pp. SO CODEN: PIXXD2 DT Patent LA English FAN.CNT 1 APPLICATION NO. DATE PATENT NO. KIND DATE ____ WO 9842362 PΙ A119981001 WO 98-US6208 19980327 W: AU, CA, JP RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE AU 9867865 19981020 AU 98-67865 A119980327 PRAI US 97-828010 19970327 WO 98-US6208 19980327 The present invention relates to the mammalian fsh05 gene, a novel gene assocd. with bipolar affective disorder (BAD) in humans. The invention encompasses fsh05 nucleic acids, recombinant DNA mols., cloned genes or degenerate variants thereof, fsh05 gene products and antibodies directed against such gene products, cloning vectors contg. mammalian fsh05 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh05 and to using such compds. as therapeutic agents in the treatment of fsh05 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh05 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and

compns. for the treatment of these disorders.

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ANSWER 6 OF 21 CA COPYRIGHT 1999 ACS
L9
    129:286742 CA
ΑN
    Fsh16 gene and methods and compositions for the diagnosis and treatment
ΤI
of
    neuropsychiatric disorders
ΤN
    Chen, Hong; Freimer, Nelson B.
    Millenium Pharmaceuticals, Inc., USA; The Regents of the University of
PA
    California
     PCT Int. Appl., 93 pp.
SO
    CODEN: PIXXD2
     Patent
LA
    English
FAN. CNT 1
                                        APPLICATION NO. DATE
     PATENT NO.
                     KIND DATE
                                          _____
     _____
                     ____
                          _____
                           19981001
                                         WO 98-US6210
                                                           19980327
    WO 9842726
                     A1
РΤ
     W: AU, CA, JP
        RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT,
                                       US 97-828009
                                                           19970327
    US 5914394
                           19990622
                      Α
                           19981020
                                          AU 98-67867
                                                           19980327
    AU 9867867
                      A1
PRAI US 97-828009
                     19970327
                     19980327
    WO 98-US6210
    The present invention relates to the mammalian fsh16 gene, a novel gene
AB
    assocd. with bipolar affective disorder (BAD) in humans. The
    invention encompasses fsh16 nucleic acids, recombinant DNA mols., cloned
     genes or degenerate variants thereof, fsh16 gene products and antibodies
    directed against such gene products, cloning vectors contg. mammalian
    fsh16 gene mols., and hosts that have been genetically engineered to
    express such mols. The invention further relates to methods for the
     identification of compds. that modulate the expression of fsh16 and to
     using such compds. as therapeutic agents in the treatment of fsh16
     disorders and neuropsychiatric disorders. The invention also relates to
    methods for the diagnostic evaluation, genetic testing and prognosis of
     fsh16 disorders and neuropsychiatric disorders including schizophrenia,
     attention deficit disorder, a schizoaffective disorder, a bipolar
     affective disorder or a unipolar affective disorder, and to methods and
    compns. for the treatment of these disorders.
T.9
    ANSWER 7 OF 21 CA COPYRIGHT 1999 ACS
AN
    129:286740 CA
    Fsh22 gene and methods and compositions for the diagnosis and treatment
TΙ
οf
    neuropsychiatric disorders
    Chen, Hong; Freimer, Nelson B.
IN
    Millenium Pharmaceuticals, Inc., USA; The Regents of the University of
PA
    California
     PCT Int. Appl., 93 pp.
SO
     CODEN: PIXXD2
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    WO 9842723 A1
                                         WO 98-US6209 19980327
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        RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT,
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                                         AU 98-67866
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    AU 9867866
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PRAI US 97-828008
                     19970327
    WO 98-US6209
                     19980327
    The present invention relates to the mammalian fsh22 gene, a novel gene
AB
     assocd. with bipolar affective disorder (BAD) in humans. The
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invention encompasses fsh22 nucleic acids, recombinant DNA mols., cloned genes or degenerate variants thereof, fsh22 gene products and antibodies directed against such gene products, cloning vectors contg. mammalian fsh22 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh22 and to using such compds. as therapeutic agents in the treatment of fsh22 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh22 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compns. for the treatment of these disorders.

- L9 ANSWER 8 OF 21 CA COPYRIGHT 1999 ACS
- AN 129:271555 CA
- TI Fsh15w6 gene and methods and compositions for the diagnosis and treatment of neuropsychiatric disorders
- IN Chen, Hong; Freimer, Nelson B.
- PA Millenium Pharmaceuticals, Inc., USA; The Regents of the University of California
- SO PCT Int. Appl., 94 pp. CODEN: PIXXD2
- DT Patent
- LA English
- FAN.CNT 1

	PATENT NO.				KIND		DATE			APPLICATION NO.					DATE			
PI	WO 9842724 W: AU, CA,			A1		19981001			WO 98-US6211					19980327				
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			AT,		CH,	DE,	DK,	ES,	FI,	FR,	GB,	GR,	ΙE,	IT,	LU,	MC,	NL,	PT,
SE												•	•				·	·
	US	US 5866412			A		19990202			US 97-828007			19970327					
	AU 9867868			A.	1	19981020		AU 98-67868				19980327						
PRAI	US	JS 97-828007			19970327													
	WO 98-US6211			19980327														

AB The present invention relates to the mammalian fsh15w6 gene, a novel gene assocd. with **bipolar** affective disorder (BAD) in humans. The invention encompasses fsh15w6 nucleic acids, recombinant DNA mols.,

genes or degenerate variants thereof, fsh15w6 gene products and antibodies

directed against such gene products, cloning vectors contg. mammalian fsh15w6 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh15w6 and to using such compds. as therapeutic agents in the treatment of fsh15w6 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh15w6 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compns. for the treatment of these disorders.

- L9 ANSWER 9 OF 21 CA COPYRIGHT 1999 ACS
- AN 129:1406 CA
- TI Chromosomal markers and diagnostic tests for manic-depressive illness
- IN Detera-Wadleigh, Sevilla D.; Gershon, Elliot S.; Badner, Judith A.;
 Goldin, Lynn R.; Berrettini, Wade H.; Yoshikawa, Takeo; Sanders, Alan R.;
 Esterling, Lisa E.
- PA United States Dept. of Health and Human Services, USA; Detera-Wadleigh, Sevilla D.; Gershon, Elliot S.; Badner, Judith A.; Goldin, Lynn R.; Berrettini, Wade H.; Yoshikawa, Takeo; Sanders, Alan R.; Esterling, Lisa E.

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SO
      PCT Int. Appl., 119 pp.
      CODEN: PIXXD2
DT
      Patent
LA
     English
FAN.CNT 1
    PATENT NO.
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                       KIND DATE
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                                            Wo 97-US19381
                       A1
                             19980507
                                                               19971028
ΡI
     WO 9818963
        W. AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR,
              KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ,
              PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG,
              US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG
                       AA 19980507
                                             CA 97-2241855
     CA 2241855
                                                               19971028
     AU 9851509
                        A1
                              19980522
                                             AU 98-51509
                                                               19971028
PRAI US 96-29278
                       19961028
                       19971028
     WO 97-US19381
     Methods and compns. are provided for detg. a genotype assocd. with
AB
     increased susceptibility to manic-depressive illness. The
     genotype is detd. using markers for a region of chromosome
     18 exhibiting linkage disequil. with manic-depressive
     illness. The invention also provides for a novel myo-inositol
     monophosphatase protein encoded for on chromosome 18.
     Using direct cDNA selection and phys. mapping by PCR, 25 novel,
     chromosome 18-specific cDNAs expressed in infant brain
     have been identified and positionally cataloged. A cDNA for a gene
     assocd. with manic-depression was identified. Based on sequence
     homol. and presence of protein motifs, the gene is proposed to encode
     myo-inositol monophosphatase. The promoter region of the gene was also
     isolated and sequenced.
1.9
     ANSWER 10 OF 21 CA COPYRIGHT 1999 ACS
AN
     128:253385 CA
TI
     Genomic screening in manic-depressive disorder
AII
     Verheyen, Geert R.; Van Broeckhoven, Christine
CS
     Laboratory of Neurogenetics, Flanders Interuniversity Institute for
     Biotechnology (VIB), Department of Biochemistry, Born-Bunge Foundation
     (BBS), University of Antwerp (UIA), Antwerp, B-2610, Belg.
SO
     Wenner-Gren Int. Ser. (1998), 69 (Genetics and Psychiatric Disorders),
     147-163
     CODEN: WGISEA; ISSN: 1356-0409
PB
     Elsevier Science Ltd.
DT
     Journal; General Review
LA
     English
     A review with .apprx.50 refs., providing an overview of the results of
AΒ
the
     linkage studies in several bipolar disorder families performed
     in the authors' lab. The authors have mostly found neg. linkage results.
     However, Xq27-q28 could not be excluded and small, pos. LOD scores are
     obtained. Suggestive LOD scores were also found for linkage to
     18q22.3-q23.
L9
     ANSWER 11 OF 21 CA COPYRIGHT 1999 ACS
ΑN
     128:176630 CA
TI
     Rapid cloning of expanded trinucleotide repeat sequences from genomic DNA
     Koob, Michael D.; Benzow, Kellie A.; Bird, Thomas D.; Day, John W.;
AU.
     Moseley, Melinda L.; Ranum, Laura P. W.
CS
     Dep. Neurol., Univ. Minnesota, Minneapolis, MN, 55455, USA
     Nat. Genet. (1998), 18(1), 72-75
SO
     CODEN: NGENEC; ISSN: 1061-4036
PB
     Nature America
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Journal

DΨ

LA English

AB Trinucleotide repeat expansions have been shown to cause a no. of neurodegenerative diseases. A hallmark of most of these diseases is the presence of anticipation, a decrease in the age at onset in consecutive generations due to the tendency of the unstable trinucleotide repeat to lengthen when passed from one generation to the next. The involvement of trinucleotide repeat expansions in a no. of other diseases - including familial spastic paraplegia, schizophrenia, bipolar affective disorder and spinocerebellar ataxia type 7 (SCA7) - is suggested both by the presence of anticipation and by repeat expansion detection (RED)

of genomic DNA samples. The involvement of trinucleotide expansions in these diseases, however, can be conclusively confirmed only by the isolation of the expansions present in these populations and detailed anal. to assess each expansion as a possible pathogenic mutation. We describe a novel procedure for quick isolation of expanded trinucleotide repeats and the corresponding flanking nucleotide sequence directly from small amts. of genomic DNA by a process of Repeat Anal., Pooler Isolation and Detection of individual clones contg. expanded trinucleotide repeats (RAPID cloning). We have used this technique to clone the pathogenic

SCA7

anal.

CAG expansion from an archived DNA sample of an individual affected with ataxia and retinal degeneration.

- L9 ANSWER 12 OF 21 CA COPYRIGHT 1999 ACS
- AN 128:10756 CA
- TI Genomic structure and chromosomal localization of a human myo-inositol monophosphatase gene (IMPA)
- AU Sjoholt, Gry; Molven, Anders; Lovlie, Roger; Wilcox, Andrea; Sikela, James

M.; Steen, Vidar M.

- CS Dr. Einar Martens' Research Group for Bioogical Psychiatry, Center for Molecular Medicine, Haukeland University Hospital, Bergen, N-5021, Norway
- SO Genomics (1997), 45(1), 113-122 CODEN: GNMCEP; ISSN: 0888-7543
- PB Academic
- DT Journal
- LA English
- AB Manic-depressive illness is a serious psychiatric disorder that in many, but far from all, patients can be treated with lithium. The main

causes for discontinuation of lithium therapy are unpleasant or serious side effects and lack of response. The reason for the striking variation in clin. efficacy of lithium treatment among bipolar patients is not known. The enzyme myo-inositol monophosphatase (IMPase) has been postulated as a target for the mood-stabilizing effects of lithium, but variation in the coding region of the human IMPA gene encoding IMPase activity has not been obsd. in manic-depressive patients (Steen et al., Pharmacogenetics, 1996, 6, 113-116). It is nevertheless conceivable that polymorphisms or mutations in the noncoding regions of this gene could influence the lithium response in psychiatric patients. As a first step in investigating this possibility, we here report the genomic structure of the human IMPA gene. The gene is composed of at least nine exons and covers more than 20 kb of sequence on chromosome 8q21.13-q21.3. In the 3'-untranslated part of the gene, we obsd. a polymorphism (a G to A transition) and also two short sequences similar

to

the inositol/cholin-responsive element consensus. Finally, we postulate that two addnl. IMPA-like transcripts originate from the human genome,

one

from a position close to IMPA itself on chromosome 8 and the other from chromosome 18p. Our data may contribute to the identification of genetic factors involved in the pathogenesis and detn. of treatment response in manic-depressive illness.

ANSWER 13 OF 21 CA COPYRIGHT 1999 ACS L9 128:842 CA AN A novel, heritable, expanding CTG repeat in an intron of the SEF2-1 gene ΤI on chromosome 18q21.1 ΑU Breschel, T. S.; McInnis, M. G.; Margolis, R. L.; Sirugo, G.; Corneliussen, B.; Simpson, S. G.; McMahon, F. J.; MacKinnon, D. F.; Xu, J. F.; Pleasant, N.; Huo, Y.; Ashworth, R. G.; Grundstrom, C.; Grundstrom, T.; Kidd, K. K.; DePaulo, J. R.; Ross, C. A. George Browne Genet. Lab., Dep. Psychiatry Behav. Sci., Johns Hopkins CS Univ. Sch. Med., Baltimore, MD, USA SO Hum. Mol. Genet. (1997), 6(11), 1855-1863 CODEN: HMGEE5; ISSN: 0964-6906 PB Oxford University Press DTJournal LAEnglish There are currently 13 diseases known to be caused by unstable triplet AΒ repeat mutations; however, there are some instances (as with FRAXF and FRA16) when these mutations appear to be asymptomatic. In a search for polymorphic CTG repeats as candidate genes for bipolar disorder, we screened a genomic human chromosome 18-specific library and identified a 1.6 kb clone (7,6A) with a CTG24 repeat that maps to 18q21.1. The CTG repeat locus, termed CTG18.1, is located within an intron of human SEF2-1, a gene encoding a basic helix-loop-helix DNA binding protein involved in transcriptional regulation. The CTGn repeat is highly polymorphic and very enlarged alleles, consistent with expansions of up to CTG2100, were identified. PCR and Southern blot anal. in pedigrees ascertained for a Johns Hopkins University bipolar disorder linkage study and in CEPH ref. pedigrees revealed a tripartite distribution of CTG18.1 alleles with stable alleles (CTG10-CTG37), moderately enlarged and unstable alleles (CTG53-CTG250), and very enlarged, unstable alleles (CTG800-CTG2100). Moderately enlarged alleles were not assocd. with an abnormal phenotype and have a combined enlarged allele frequency of 3% in the CEPH and bipolar populations. Very enlarged alleles, detectable only by Southern blot anal. of genomic digests, have thus far been found in only three individuals from our bipolar pedigrees, and to date, have not been found in any of the CEPH ref. pedigrees. These enlarged alleles may arise, at least in part, via somatic mutation. L9 ANSWER 14 OF 21 CA COPYRIGHT 1999 ACS AN127:327441 CA ΤI Methods for detecting bipolar mood disorder susceptibility locus on human chromosome (18q) Friemer, Nelson B.; heon, Pedro; Reus, Victor I.; Sandkuijl, Lodewijk A.; TN Barondes, Samuel H. PA Regents of the University of California, USA; Friemer, Nelson B.; Leon, Pedro; Reus, Victor I.; Sandkuijl, Lodewijk A.; Barondes, Samuel H. PCT Int. Appl., 51 pp. SO CODEN: PIXXD2 DT Patent English LAFAN.CNT 1 KIND DATE PATENT NO. APPLICATION NO. DATE ~ - - -A1 19971009 WO 9737043 PI WO 97-US4904 19970327 W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ,

VN, YU, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN,

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ML, MR, NE, SN, TD, TG
                                                                 19970327
                              19971009
                                              CA 97-2247996
                        AA
     CA 2247996
                                                                 19970327
                                              AU 97-24238
     AU 9724238
                        A1
                              19971022
                                              WO 97-US14892
                                                                19970822
                              19980226
                        A1
     WO 9807887
            AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
             PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ,
         VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA,
              GN, ML, MR, NE, SN, TD, TG
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                                              AU 97-41604
     AU 9741604
                        A1 19980306
PRAI US 96-14498
                        19960329
                       19960823
     US 96-23438
                       19970327
     WO 97-US4904
                       19970822
     WO 97-US14892
     The present invention is directed to methods of detecting the presence of
AB
     a bipolar mood disorder susceptibility locus in an individual,
     comprising analyzing a sample of DNA for the presence of a DNA
     polymorphism on the long arm of chromosome 18 between
     markers D18S469 and D18S554, wherein the DNA polymorphism is assocd. With
     a form of bipolar mood disorder (BP). The invention for the
     first time provides strong evidence of a susceptibility gene for BP that
     is located in the 18q22-q23 region of the long arm of chromosome
          The disclosure describes the use of linkage anal. and genetic
     markers in the 18q22-q23 region to fine map the region and the use of
     genetic markers to genetically diagnose (genotype) BP in individuals, to
     confirm phenotypic diagnoses of BP, to det. appropriate treatments for
     patients with particular genotypic subtypes. Isolated polynucleotides
     useful for genetic linkage anal. of BP-I and methods for obtaining such
     isolated polynucleotides are also described. In screening for a BP
     susceptibility locus, only those individuals with the most severe and
     clin. distinctive form of BP were considered as affected. Two large
     pedigrees were selected from a genetically homogeneous population, that
of
     the Central Valley of Costa Rica. The entire human genome was screened
      for linkage using mapped microsatellite markers and a model for genetic
      anal. in which most of the linkage information derived from affected
      individuals. Three lines of evidence supported the localization of a BP
      susceptibility locus to 18q22-q23: assocn. anal., linkage anal., and
     direct observation of a conserved marker haplotype.
     ANSWER 15 OF 21 CA COPYRIGHT 1999 ACS
L9
     126:5800 CA
      A complete genome screen for genes predisposing to severe bipolar
TT
      disorder in two Costa Rican pedigrees
     McInnes, L. Alison; Escamilla, Michael A.; Service, Susan K.; Reus,
ΑU
Victor
      I.; Leon, Pedro; Silva, Sandra; Rojas, Eugenia; Spesny, Mitzi; Baharloo,
      Siamak; et al.
      Neurogenet. Lab., Univ. California, San Francisco, CA, 94143, USA
CS
     Proc. Natl. Acad. Sci. U. S. A. (1996), 93(23), 13060-13065
SO
      CODEN: PNASA6; ISSN: 0027-8424
      National Academy of Sciences
PB
      Journal
DT
      English
LA
      Bipolar mood disorder (BP) is a debilitating syndrome
AΒ
      characterized by episodes of mania and depression. We designed a
      multistage study to detect all major loci predisposing to severe BP
      (termed BP-I) in two pedigrees drawn from the Central Valley of Costa
      Rica, where the population is largely descended from a few founders in
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16th-18th centuries. We considered only individuals with BP-I as affected

and screened the genome for linkage with 473 microsatellite markers. We used a model for linkage anal. that incorporated a high phenocopy rate

and

a conservative est. of penetrance. Our goal in this study was not to establish definitive linkage but rather to detect all regions possibly harboring major genes for BP-I in these pedigrees. To facilitate this aim, we evaluated the degree to which markers that were informative in

our

data set provided coverage of each genome region; we est. that at least 94% of the genome has been covered, at a predesignated threshold detd. through prior linkage simulation analyses. We report here the results of our genome screen for BP-I loci and indicate several regions that merit further study, including segments in 18q, 18p, and 11p, in which suggestive lod scores were obsd. for two or more contiguous markers. Isolated lod scores that exceeded our thresholds in one or both families also occurred on chromosomes 1, 2, 3, 4, 5, 7, 13, 15, 16, and 17. Interesting regions highlighted in this genome screen will be followed up using linkage disequil (LD) methods.

L9 ANSWER 16 OF 21 CA COPYRIGHT 1999 ACS

AN 125:192566 CA

TI No association between **chromosome-18** markers and lithium-responsive affective disorders

AU Turecki, Gustavo; Alda, Martin; Grof, Paul; Grof, Eva; Martin, Rory; Cavazzoni, Patrizia A.; Duffy, Anne; Maciel, Patricia; Rouleau, Guy A.

CS Centre Research Neuroscience, Montreal General Hospital, Montreal, PQ,

нзн

1A4, Can.

SO Psychiatry Res. (1996), 63(1), 17-23 CODEN: PSRSDR; ISSN: 0165-1781

DT Journal

LA English

AB An allelic assocn. study of excellent responders to lithium was conducted with a candidate gene (Golf, a G-protein receptor gene) and five other chromosome-18p markers. Golf is of special interest because it maps to a region of chromosome 18 where two independent groups (Berrettini et al., 1994; Stine et al., 1995) have found linkage to bipolar disorder. It has been proposed that G proteins are involved in the pathogenesis of bipolar disorder, and lithium, an effective prophylactic agent, is known to impair G-protein activation. To reduce heterogeneity - a common obstacle to genetic investigation - only patients who showed excellent response to lithium prophylaxis were studied. Fifty-five genetically unrelated excellent responders to

lithium

prophylaxis were compared with 94 normal subjects of similar ethnic background. The groups did not differ in either allele or genotype frequency for the tested markers. The data do not support the hypothesis that the tested loci confer a major susceptibility for affective disorders.

L9 ANSWER 17 OF 21 CA COPYRIGHT 1999 ACS

AN 125:2666 CA

TI Genetic mapping using haplotype, association and linkage methods suggests a locus for severe bipolar disorder (BPI) at 18g22-g23

AU Freimer, Nelson B.; Reus, Victor I.; Escamilla, Michael A.; McInnes, L. Alison; Spesny, Mitzi; Leon, Pedro; Service, Susan K.; Smith, Lauren B.; Silva, Sandra; et al.

CS Neurogenetics Laboratory, Univ. of California San Francisco, San Francisco, CA, 94143, USA

SO Nat. Genet. (1996), 12(4), 436-441 ... CODEN: NGENEC; ISSN: 1061-4036

DT Journal

LA English

AB Manic-depressive illness, or bipolar disorder (BP), is characterized by episodes of elevated mood (mania) and depression1. We



designed a multistage study in the genetically isolated population of the Central Valley of Costa Rica2,3 to identify genes that promote susceptibility to severe BP (termed BPI), and screened the genome of two Costa Rican BPI pedigrees (McInnes et al., submitted). We considered only individuals who fulfilled very stringent diagnostic criteria for BPI to be affected. The strongest evidence for a BPI locus was obsd. in 18q22-q23. We tested 16 addnl. markers in this region and seven yielded peak lod scores over 1.0. These suggestive lod scores were obtained over a far greater chromosomal length (about 40 cM) than in any other genome region. This localization is supported by marker haplotypes shared by 23 of 26 BPT affected individuals studied. Addnl., marker allele frequencies over portions of this region are significantly different in the patient sample from those of the general Costa Rican population. Finally, we performed an anal. which made use of both the evidence for linkage and for assocn. in 18q23, and we obsd. significant lod scores for two markers in this region. L9 ANSWER 18 OF 21 CA COPYRIGHT 1999 ACS NA125:2629 CA TΙ Analysis of chromosome 18 DNA markers in multiplex pedigrees with manic depression ΑIJ Coon, Hilary; Hoff, M.; Holik, J.; Hadley, D.; Fang, N.; Reimherr, F.; Wender, P.; Byerley, William CS Medical School, University Utah, Salt Lake City, UT, 84132, USA Biol. Psychiatry (1996), 39(8), 689-696 SO CODEN: BIPCBF; ISSN: 0006-3223 DTJournal LAEnglish AΒ Six pedigrees segregating manic-depressive illness (MDI) were analyzed for linkage to 21 highly polymorphic microsatellite DNA markers on chromosome 18. These markers span almost the entire length of the chromosome, and gaps between markers are less than 20 cM. In particular, we analyzed several markers localizing to the pericentromeric region of chromosome 18 which generated lod scores suggestive of linkage in an independent study. Lod score anal. was performed and results were examd. by family. One region produced pos. lod scores, though at 18q23 and not in the pericentromeric region. We addnl. used two nonparametric methods because the true mode of transmission of MDI is unknown; results were again somewhat suggestive for markers in the region of 18q23 but not in the pericentromeric region. L9 ANSWER 19 OF 21 CA COPYRIGHT 1999 ACS 124:334471 CA AΝ Linkage analysis of families with bipolar illness and TIchromosome 18 markers De bruyn, An; Souery, Daniel; Mendelbaum, Karine; Mendlewicz, Julien; Van ΑIJ Broeckhoven, Christine Neurogenetics Laboratory, University Antwerp (UIA), Antwerpe, B-2610, CS SO Biol. Psychiatry (1996), 39(8), 679-688 CODEN: BIPCBF; ISSN: 0006-3223 DT Journal LA English AΒ Linkage of bipolar (BP) illness with chromosome 18 markers located at 18p11 was recently reported. A possible role for chromosome 18 in the etiol. of BP illness was implicated previously by the finding in three unrelated patients of a

chromosome with breakpoints and deleted segments at 18pter-p11 and

18q23-qter. To test the potential importance of a gene defect on chromosome 18 in our material, we examd. linkage with chromosome 18 markers in two families with multiple patients with BP illness or BP spectrum disorders. Fourteen simple tandem

repeat polymorphisms were used located in the chromosomal region 18p11 to 18q23 and sepd. by distances of approx. 10 cM on the genetic map. In one family linkage to **chromosome 18** could not be excluded.

Linkage and segregation anal. in the family suggests that the $12\mbox{-cM}$ region

between D18S51 and D18S61 located at 18q21.33-q23 may contain a candidate gene for BP illness.

- L9 ANSWER 20 OF 21 CA COPYRIGHT 1999 ACS
- AN 124:108441 CA
- TI Evidence for linkage of **bipolar** disorder to **chromosome**18 with a parent-of-origin effect
- AU Stine, O. Colin; Xu, Jianfeng; Koskela, Rebecca; McMahon, Francis J.; Gschwend, Michele; Friddle, Carl; Clark, Chris D.; Mclnnis, Melvin G.; Simpson, Sylvia G.; et al.
- CS School Medicine, Johns Hopkins University, Baltimore, USA
- SO Am. J. Hum. Genet (1995), 57(6), 1384-94 CODEN: AJHGAG; ISSN: 0002-9297
- DT Journal
- LA English

on

AB A susceptibility gene on chromosome 18 and a parent-of-origin effect have been suggested for bipolar affective disorder (BPAD). We have studied 28 nuclear families selected for apparent unilinear transmission of the BPAD phenotype, by using 31 polymorphic markers spanning chromosome 18. Evidence for linkage was tested with affected-sib-pair and LOD score methods under

for linkage was tested with affected-sib-pair and LOD score methods under two definitions of the affected phenotype. The affected-sib-pair analyses

indicated excess allele sharing for makers on 18p within the region reported previously. The greatest sharing was at D18S37: 64% in **bipolar** and recurrent unipolar (RUP) sib pairs (P = .0006). In addn., excess sharing of the paternally, but not maternally, transmitted alleles was obsd. at three markers on 18q: at D18S41, 51 **bipolar** and RUP sib pairs were concordant for paternally transmitted alleles, and 21 pairs were discordant (P = .0004). The evidence for linkage to loci

both 18p and 18q was strongest in the 11 paternal pedigrees, i.e., those in which the father or one of the father's sibs is affected. In these pedigrees, the greatest allele sharing (81%; P = .00002) and the highest LOD score (3.51; .THETA. = 0.0) were obsd. at D18S41. Our results provide

further support for linkage of BPAD to chromosome 18 and the first mol. evidence for a parent-of-origin effect operating in this disorder. The no. of loci involved, and their precise location, require further study.

- L9 ANSWER 21 OF 21 CA COPYRIGHT 1999 ACS
- AN 121:55150 CA
- TI Chromosome 18 DNA markers and manic -depressive illness: evidence for a susceptibility gene
- AU Berrettini, Wade H.; Ferraro, Thomas N.; Goldin, Lynn R.; Weeks, Daniel E.; Detera-Wadleigh, Sevilla; Nurnberger, John I., Jr.; Gershon, Elliot
- S.
 CS Dep. Psychiatry and Human Behavior, Thomas Jefferson Univ., Philadelphia,
 PA, 19107, USA
- SO Proc. Natl. Acad. Sci. U. S. A. (1994), 91(13), 5918-21 CODEN: PNASA6; ISSN: 0027-8424
- DT Journal
- LA English
- AB In the course of a systematic genomic survey, 22 manic

-depressive (bipolar) families were examd. for linkage to 11 chromosome 18 pericentromeric marker loci, under dominant and recessive models. Overall logarithm of odds score anal. for the pedigree series was not significant under either model, but several families yielded logarithm of odds scores consistent with linkage under dominant or recessive models. Affected sibling pair anal. of these data yielded evidence for linkage (P < 0.001) at D18S21. Affected pedigree member anal. also suggests linkage, with multilocus results for five loci giving P < 0.0001 and P = 0.0007 for weighting functions f(p) = 1 and 1/.sqroot.p, resp., where p is the allele frequency. These results imply a susceptibility gene in the pericentromeric region of chromosome 18, with a complex mode of inheritance. Two plausible candidate genes, a ACTH receptor and the .alpha. subunit of a GTP binding protein, have been localized to this region.

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L10
      ANSWER 1 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
AN
      99-04694 BIOTECHDS
TT
      New isolated fsh05 gene;
         associated with human bipolar affective disorder, useful for
         drug screening, diagnosis, therapy, mapping and DNA polymorphism
         identification of central nervous system disease, e.g. stroke
ΔIJ
      Chen H; Freimer N B
PA
      Millennium-Pharm.; Univ.California
LO
      Cambridge, MA, USA; Oakland, CA, USA.
      WO 9904825 4 Feb 1/999
PΤ
AΙ
      WO 98-US15183 22 Jul 1998
PRAI
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DT
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T.A
      English
OS
      WPI: 99-142616 [12]
L10
      ANSWER 2 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
AN
      99-02653 BIOTECHDS
TI
      New isolated human fsh05 gene;
         recombinant fsh05 gene, protein and antibody used to diagnose and
         treat neurop/sychiatric disorder
ΑU
      Chen H; Freimer N B
      Millennium-Pharm.; Univ.California
PA
      Cambridge, /MA, USA; Oakland, CA, USA.
LO
      WO 9842362 1 Oct 1998
PΤ
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AI
PRAI
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DT
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      WPI: 99-070062 [06]
OS
T_110
      ANSWER 3 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
      99-02055 BIOTECHDS
ΑN
ΤI
      New isolated human fsh16 gene;
         protein and antibody used for neuropsychiatric condition diagnosis,
         therapy and drug screening, and to identify fsh16 gene polymorphism
ΑU
      Chen H; Freimer N B
PA
      Millennium-Pharm.; Univ.California
      Cambridge, MA, USA; Oakland, CA, USA. WO 9842726 1 Oct 1998
LO
PΙ
      WO 98-US6210 27 Mar 1998
AΤ
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      ANSWER 4 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
T<sub>1</sub>1.0
      99-00102 BIOTECHDS
MΑ
TI
      New isolated human fsh15w6 gene;
         recombinant protein and encoding DNA for use in neuropsychiatric
         disease diagnosis, therapy and drug screening
ΑU
      Chen H; Freimer N B
      Millennium-Pharm.; Univ.Çalifornia
PA
LO
      Cambridge, MA, USA; Oakland, CA, USA.
      WO 9842724 1 Oct 1998
PΙ
      WO 98-US6211 27 Mar 1998
ΑI
PRAI
     US 97-828007 27 Már 1997
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OS
      WPI: 98-542273 [46]
L10
      ANSWER 5 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
AN
      99-00101 BIOTECHDS
TI
      New isolated human fsh22 gene;
         recombinant protein and encoding DNA for use in neuropsychiatric
         disease diagnosis, therapy and drug screening
ΑU
      Chen H; Freimer N B
      Millennium-Pharm.; Univ.California
PA
      Cambridge, MA, USA; Oakland, CA, USA.
LO
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PΙ
      WO 98-US6209 27 Mar 1998
AΙ
PRAI
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LA
      WPI: 98-542272 [46]
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      ANSWER 6 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
L10
ΑN
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TI
      New isolated IMP.18p myo-inositol-monophosphatase;
         human myo-inositol-monophosphatase gene-specific DNA primer
         construction, antibody and antisense DNA, used for manic
         -depressive illness susceptibility determination or therapy
ΑU
      Detera-Wadleigh S D; Gershon E S; Badner J A; Goldin L R; Berrettini W
Η:
      Yoshikawa T; Sanders A R; Esterling L E
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AN
      98-00993 BIOTECHDS
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      Medical methods relating to bipolar mood disorder;
         genotype analysis for use in diagnosis
ΑU
      Friemer N B; Leon P; Reus V I; Sandkuijl L A; Barondes S H
     Univ.California
PΑ
LO
      Oakland, CA, USA.
     WO 9737043 9 Oct 1997
PΤ
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PRAI US 96-23438 23 Aug 1996; US 96-14498 29 Mar 1996
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F. Baas, University of Amsterdam, Academic Medical Center, PO Box 22700,

J.M.N.; Schalling M.; De Belleroche J.; Baas F.

1100DE Amsterdam, Netherlands. f.baas@amc.uva.nl

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L11 ANSWER 11 OF 74 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
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     Closing in on genes for manic-depressive illness and
     schizophrenia.
     Gershon E.S.; Badner J.A.; Goldin L.R.; Sanders A.R.; Cravchik A.;
AU
     Detera-Wadleigh S.
     Dr. E.S. Gershon, Clinical Neurogenetics Branch, National Institute of
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     Mental Health, National Institutes of Health, Bethesda, MD 20892-1274,
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     bipolar disorder.
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     Gershon E.S.; Moskowitz M.T.; Detera-Wadleigh S.D.
     L.E. Esterling, Clinical Neurogenetics Branch, National Institute of
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     Bethesda, MD 20892-1274, United States
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     Linkage of bipolar affective disorder to chromosome
     18 markers in a new pedigree series.
     McMahon F.J.; Hopkins P.J.; Xu J.; McInnis M.G.; Shaw S.; Cardon L.;
ΑU
     Simpson S.G.; MacKinnon D.F.; Stine O.C.; Sherrington R.; Meyers D.A.;
     DePaulo J.R.
     Dr. F.J. McMahon, Meyer 3-181, 600 North Wolfe Street, Baltimore, MD
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     21287-7381, United States. fmcm@welchlink.welch.jhu.edu
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     ANSWER 14 OF 74 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
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     Model-free age-of-onset methods applied to the linkage of bipolar
     disorder.
     Zhu X.; Olson J.M.; Schnell A.H.; Elston R.C.
ΑIJ
     Dr. R.C. Elston, Dept. of Epidemiology/Biostatistics, Case Western
CS
Reserve
     University, 2500 MetroHealth Drive, Cleveland, OH 44109, United States
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AN
ΤТ
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ΑU
     Wyszynski D.F.; Doetsch J.P.; Pugh E.W.; Bailey-Wilson J.E.
     Dr. D.F. Wyszynski, NIH, NHGRI, CIDR, 333 Cassell Dr., Baltimore, MD
CS
     21224, United States
     Genetic Epidemiology, (1997) 14/6 (705-710).
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     Probabilistic diagnosis in linkage analysis of bipolar disorder:
     Putting weights on the fringe.
     Van Eerdewegh P.; Santangelo S.L.; Lee H.; Laird N.M.; Blacker D. S.L. Santangelo, Department of Psychiatry, Tufts/New England Medical
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     Center, NEMC Box 1007, 750 Washington Street, Boston, MA 02111, United
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TΙ
     Modeling the phenotype in parametric linkage analysis of bipolar
     disorder.
ΑIJ
     Turecki G.; Rouleau G.; Morgan K.
CS
     G. Turecki, Centre for Research in Neuroscience, McGill University,
     Montreal General Hospital, 1650 Cedar Ave., Montreal, Que. H3G 1A4,
Canada
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     Genetic Epidemiology, (1997) 14/6 (687-691).
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- LAEnglish
- ST English
- ANSWER 18 OF 74 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V. L11
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- ΑU Simonsen K.L.; Kaplan N.L.; Martin E.R.
- K.L. Simonsen, Department of Statistics, North Carolina State University, CS Raleigh, NC 27695-8203, United States
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- ΑN 1998018191 EMBASE
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- ΑU Schnell A.H.; Karunaratne P.M.; Witte J.S.; Dawson D.V.; Elston R.C.
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- ΑIJ Margaritte-Jeannin P.; Babron M.-C.; Genin E.; Eichenbaum-Voline S.; Clerget-Darpoux F.
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     chromosome 18.
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     bipolar disease.
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Universitat Munchen, Klinikum rechts der Isar, Ismaninger Strasse 22,

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     identify a major susceptibility locus for bipolar affective
     disorder in the Old Order Amish
     Pauls, D.L.; Ott, J.; Paul, S.M.; Allen, C.R.; Fann, C.S.J.; Carulli,
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     Univ., 1025 Walnut St., 312 College, Philadelphia, PA 19107, USA
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     Inst. for Basic Psychiatric Res., Dep. Psychiatric Demography,
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     Hosp., Aarhus, Denmark
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